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Differential family and peer environmental factors are related to severity and comorbidity in children with ADHD

C. J. M. Buschgens[1], M. A. G. van Aken[2], S. H. N. Swinkels[1], M. E. Altink[1], E. A. Fliers[1], N. N. J. Rommelse[3], R. B. Minderaa[4], J. A. Sergeant[3], S. V. Faraone[5], J. K. Buitelaar[1]

[1] Department of Child and Adolescent Psychiatry, Radboud University Nijmegen Medical Centre, Nijmegen, The Netherlands
[2] Department of Developmental Psychology, Utrecht University, Utrecht, The Netherlands
[3] Department of Psychology, Vrije Universiteit, Amsterdam, The Netherlands
[4] University Center of Child and Adolescent Psychiatry, Groningen, The Netherlands
[5] Departments of Psychiatry and of Neuroscience and Physiology, SUNY Upstate Medical University, Syracuse, NY, USA

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Summary. Behavioral genetic studies imply that salient environmental influences operate *within* families, making siblings in a family different rather than similar. This study is the first one to examine differential sibling experiences (as measured with the Sibling Inventory of Differential Experience) and its effect on behavioral outcomes within ADHD families. Subjects were 45 Dutch ADHD probands and their unaffected siblings ($n = 45$) aged 10–18 years. ADHD probands and their unaffected siblings reported differences in sibling interaction, parental treatment, and peer characteristics. These nonshared environmental influences were related to both the severity of ADHD symptoms as well as to comorbid problem behaviors. These findings suggest that environmental influences that operate *within* ADHD families appear relevant to the severity of problem behaviors of ADHD children and their siblings.

Keywords: ADHD; comorbidity; nonshared environment; discordance; sibling pair

Introduction

Attention-deficit/hyperactivity disorder (ADHD; APA 1994) is a common neuropsychiatric disorder, which is characterized by a chronic pattern of inattention, impulsivity, and hyperactivity, that affects 8–12% of children worldwide (Faraone et al. 2003). Twin studies consistently indicate that the development and severity of ADHD is due to substantial genetic influences, few shared environmental influences and small-to-moderate nonshared environmental influences (Waldman and Gizer 2006). In the present study we will investigate nonshared environmental influences in ADHD by comparing ADHD probands and their unaffected biological siblings. Nonshared environmental influences are defined as aspects of the environment experienced differently by siblings in the same family (Daniels and Plomin 1985). These nonshared influences, which have also been referred to as within-family, differential, or unique influences, are considered to be crucial for understanding environmental influences on individual development (Dunn and Plomin 1990; Pike and Plomin 1996; Plomin et al. 2001).

Though considerable progress has been made in the genetics of ADHD (Faraone et al. 2005; Waldman and Gizer 2006), less focus has been given to identifying environmental risk factors that are associated with ADHD. Biological factors, including low birth weight, pregnancy and delivery complications, maternal smoking and alcohol exposure during pregnancy, and lead contamination, have repeatedly been proposed as contributors to ADHD (Banerjee et al. 2007). In addition, severity and comorbidity in children with ADHD have been linked to several adverse psychosocial influences (Schachar and Tannock 1995; Johnston and Mash 2001; Cohen et al. 2002), like divorce and remarriage, disrupted parent-child relationships (including physical abuse), parenting stress, poverty, unemployment and parental psychopathology. Furthermore, an aggregation of Rutter's indicators of family adversity also proved to be

Correspondence: Cathelijne J. M. Buschgens, Department of Child and Adolescent Psychiatry, Radboud University Nijmegen Medical Centre, P.O. Box 9101, HP 966, 6500 HB Nijmegen, The Netherlands
e-mail: c.buschgens@psy.umcn.nl

relevant for ADHD and its comorbidities (Biederman et al. 1995, 2002; Counts et al. 2005). However, it should be borne in mind that most family studies and Rutter's index of adversity are based on differences between families, which not only makes it difficult to separate the effect of environmental factors from the genetic liability imparted by the parents (Rowe 1994) but also impossible to differentiate between siblings.

Behavioral genetic research has emphasized that, when genetic effects are controlled, siblings raised within the same family are often as different from one another with regard to psychopathology and behavior problems as children raised in different families (Daniels and Plomin 1985; Daniels et al. 1985). Differential experiences unique to the individual are responsible for differences in development of child and adolescent psychopathology (Dunn and Plomin 1990; Pike and Plomin 1996; Plomin et al. 2001). It is not that family experiences are unimportant but rather that the relevant environmental influences are specific to each child, and may not generalize to the entire family (Dunn and Plomin 1990). In spite of this, few studies have actually specified nonshared environmental factors (Turkheimer and Waldron 2000), and, to our knowledge, none have focused on differences in sibling experiences within ADHD families.

Previous studies involving nonshared environmental influences have focused on other psychiatric disorders, e.g., anorexia nervosa (Murphy et al. 2000), and other forms of internalizing and externalizing behavior (e.g., Wichers et al., 2001; Asbury et al. 2003). Several studies have investigated within-family measures indirectly, e.g., by using difference-scores on variables measured individually for each sibling, while others have used the Sibling Inventory of Differential Experience (SIDE; Daniels and Plomin 1985), a measure that assesses within-family differences directly, by asking siblings about their differential experiences. A study of same-sex twins showed that direct ratings of differences in the twins' environment were associated with differences in conduct problems, whereas ad-hoc difference-scores between the ratings of each individual twin were not (Carbonneau et al. 2002). This suggests that a directly assessed contrast between siblings may be more efficient to assess aspects of within-family differences that are associated with psychopathology.

In the present sibling study, the influence of the nonshared environment is measured directly with the SIDE. Our aim is to assess the role of environmental factors, which are experienced differently by children in the same family, in the etiology of ADHD using a discordant sibling pair design. Such a design is able to provide control for part of the genetic influences (i.e., siblings share on average 50% of their genes) and for shared environmental influences (i.e., these influences make siblings in the same family more alike). This exploratory study provides an opportunity to address the following hypotheses:

(1) Sibling pairs in the same family, discordant for ADHD, experience differences in the domains of sibling interaction, parental treatment, peer relations and events specific to the individual.
(2) Such experienced differences between siblings are related to differences in their ADHD symptoms and comorbid disorders.

Material and methods

Sample collection

Subjects were Dutch participants of the IMAGE project (Brookes et al. 2006), an international collaborative study in eight European countries (Belgium, Germany, Ireland, Israel, Spain, Switzerland, the Netherlands and United Kingdom) that aims to identify genes that increase the risk for ADHD. Children (probands and siblings) could participate in IMAGE if they were aged 5–18, had an IQ $\geq$ 70, were of European Caucasian descent, and there was access to at least one biological parent for DNA collection. Entry criteria for the probands were a clinical diagnosis of DSM-

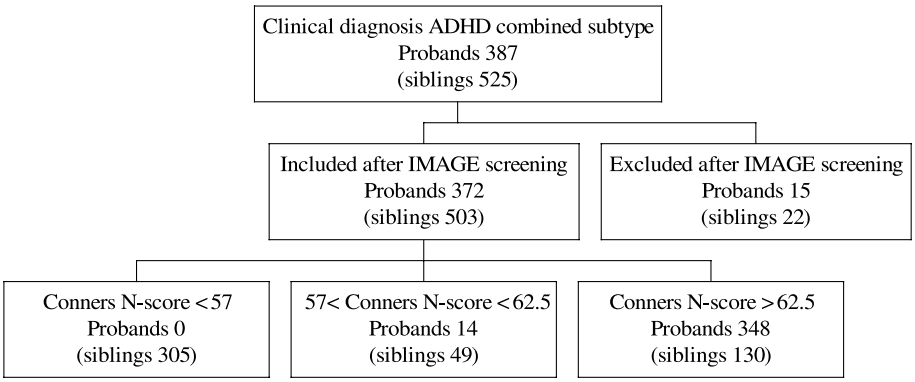

Fig. 1. Consort Flow Chart Dutch IMAGE participants

IV combined subtype of ADHD and having one or more full siblings aged 5–18 available for assessment of clinical information and DNA collection. Exclusion criteria applying to both probands and siblings included: autism, epilepsy, general learning difficulties, brain disorders and any genetic or medical disorder associated with externalizing behaviors that might mimic ADHD (see Fig. 1 for consort diagram).

After entry to the IMAGE project, the clinical diagnosis of the ADHD probands and siblings was verified with the parental account of childhood symptoms (PACS, Taylor et al. 1986). PACS is a semi-structured standardized, investigator-based interview developed as an instrument to provide an objective measure of children's behavior. Inter-rater reliability and internal consistency of the scales for behavior is good. The PACS generates information concerning symptoms in ADHD, oppositional defiant disorder, conduct disorder and emotional problems (anxiety, mood and internalizing problems). A standardized algorithm was applied to PACS to derive each of the 18 DSM-IV ADHD items, providing operational definitions for each symptom. The exact screening procedures and measures for phenotyping have been described previously (Brookes et al. 2006).

The present study involved 45 10- to 18-year-old ADHD probands and their unaffected siblings of different families, who were recruited from families referred to several participating child psychiatric outpatient clinics or from members of the Dutch Parents' Association. Wherever possible, families withdrew stimulant medication for one week prior to research assessment to allow accurate ascertainment of information on recent ADHD symptom characteristics and severity. Alternatively, we ensured as far as possible that ratings of problem behavior were based on medication free periods.

This paper reports additional data concerning family environment that have been collected in Dutch ADHD families. These data were not collected at other IMAGE sites. Not all available Dutch data, including 258 SIDEs, could be used for further analyses for two main reasons. First, only 67 families with *two* "*mutual*" *SIDEs* were included, i.e., in which (a) the ADHD proband compared himself/herself with an (un)affected sibling and (b) the (un)affected sibling compared himself/herself with the ADHD proband. Second, only 45 *discordant* sibling pairs were selected. ADHD probands were defined by an average T-score on the Conners' parent and teacher N scale (DSM-IV ADHD total symptom scores) above 62. Unaffected siblings were defined by an average T-score below 57, children with an average T-score between 57 and 62 (subthreshold) were excluded. The Medical Ethical Review Committee of Radboud University Nijmegen Medical Centre approved the study, and all parents and children from age 12 years or older signed a written informed consent form before participation in the study.

Measures

ADHD symptoms and comorbid behavior

ADHD symptoms and comorbid behaviors were assessed with two questionnaires, which were completed by the parents. The Conners' Parent Rating Scale-Revised: Long (CPRS-R:L; Conners 1997), consisting of 14 scales (80 questions), is a multimodal assessment rating scale of ADHD and related behavioral problems. In this study we focused on the Conners' subscales: DSM-IV total, Oppositional, Social problems and Anxious-Shy.

The strength and difficulties questionnaire (SDQ; Goodman 2001) is a brief measure (25 items) that assesses prosocial behavior and psychopathology of children and adolescents. In the present study the Dutch version was used (Van Widenfelt et al. 2003), containing a 3-point Likert scale. In this study, the subscales Conduct problems, Peer relationship problems and Emotional symptoms were used because of the focus of the study on comorbidity of ADHD in sibling pairs (higher scores reflect more difficulties). In order to reduce the number of dependent variables, three new variables were constructed by combining Conners' and SDQ subscales that were highly correlated. The original subscales of the Conners and SDQ were first standardized and then averaged. The new Oppositional behavior-scale consisted of Conners' Oppositional and SDQ's Conduct problems ($r = 0.81$), the Social and peer problems-scale consisted of Conners' Social problems and SDQ's Peer relationship problems ($r = 0.75$) and the Anxious and emotional problems-Scale consisted of Conners' Anxious-Shy and SDQ's Emotional symptoms ($r = 0.65$).

Differential sibling experiences

The SIDE (Daniels and Plomin 1985) is designed to assess differential experiences reported by siblings in the same family by asking each individual to compare his or her experience to those of his or her sibling on several domains. The SIDE questionnaire is appropriate for the adolescent range (i.e., 12–18 years of age) (Daniels and Plomin 1985), but is also used for children from age 10 (Pike et al. 2000). SIDE responses were based upon a scoring system with the following values: My sibling has been much more this way than I have (1); My sibling has been a bit more this way than I have (2); My sibling and I have been the same in this way (3); I have been a bit more this way than my sibling (4); I have been much more this way than by sibling (5). Four domains of differential experiences are assessed: Sibling interaction, Parental treatment, Peer characteristics, and Events specific to the individual. More details on internal consistency and sample items are presented in the Appendix.

The SIDE showed reasonable psychometric properties (Daniels and Plomin 1985). Two-week test retest reliabilities of the SIDE ranged from 0.77 to 0.93 (mean 0.84), stability across 3 years is considerable (Pike et al. 2000). For the current study, the mean Cronbach's alpha's was 0.67. The somewhat low reliability of some scales warrants caution in their interpretation. In order to compare our results to other studies, we included all scales in the analyses.

Intelligence

Pro-rated full intelligence quotient (IQ) scores were derived from four subtests of the Wechsler Intelligence Scale for Children (WISC-III^{NL}; Wechsler 2002): Vocabulary, Similarities, Block Design and Picture Completion (Sattler 2001). These subtests are known to correlate between 0.90 and 0.95 with the Full-scale IQ (Groth-Marnat 1997). Children with pro-rated IQ lower than 70 were excluded from the study.

Statistical analyses

All analyses were performed using the Statistical Package for the Social Sciences (SPSS for Windows, version 14.0). For the descriptive analyses, differences in age, sex, pro-rated IQ and behavior between the ADHD proband and unaffected sibling were examined by means of paired *t*-tests and Chi-square tests; associations between variables by means of paired sample correlations.

Repeated measures MANOVAs with differences in age, sex, and pro-rated IQ between the siblings as covariates were performed, in which the score of the ADHD probands were compared to that of their unaffected sibling on the different SIDE domains by using the sibling pair (affected vs. unaffected) as the level of the repeated measures factor (within-subject factor), and the family as the unit of analysis (between-subject factor) (Rovine 1994). If the overall main effect was significant, post-hoc tests were performed on the separate scale(s) of a domain.

Next, we investigated the relative influence of different within-family experiences to differences in siblings' symptoms or adjustment scores. Difference scores were calculated by subtracting the parent-rated behavior of the unaffected sibling from the parent-rated behavior of the proband. Although these difference scores deviated somewhat from normality, examination of the residuals did not indicate serious violation of the model assumptions. This was confirmed by the 'test of normality' (Shapiro-Wilk test was not significant), and visual inspection of the histogram and the Q–Q plot. Hierarchical regression analyses were performed with behavioral variables as dependent variables, and SIDE domains as independent variables.

Separate analyses were performed for each behavioral variable (i.e., difference scores), each SIDE domain and either proband or sibling as SIDE informant. In the first step of the regression equation, three confounders (i.e., differences in age, sex, and pro-rated IQ) were entered. In the second step the SIDE domain was entered in a stepwise model. Only SIDE domains that explained significant variance of the differences in symptoms or adjustment, over and above the effects of differences in age, sex and pro-rated IQ, will be reported by means of effect sizes for hierarchical multiple regression (f^2). This is the effect size attributable to the addition of the SIDE domain to the model, given an R^2 value for the set of confounders, and an R^2 value for the sum of the confounders and SIDE domain (Cohen 1988).

Post-hoc power analyses were performed by computing the observed power for hierarchical regression analyses, i.e., the observed power for a significance test of the addition of the SIDE domain to the hierarchical model, over and above the set of confounders, given the observed alpha level, the number of predictors in both the first and second step, the observed effect size attributable to the addition of the SIDE domain, and the sample size (Cohen 1988).

No multicollinearity was present in our data (greatest VIF value is 1.53 for peer delinquency). All testing was two-tailed and the α-level was set at 0.05.

Missing data

We employed corrected-item-mean (CIM) imputation to handle missing data at the item level (Huisman 2000). We only employed CIM with regard to SIDE data: number of missing SIDE data varied from 3% (sibling closeness) to 56% (specific events). Not all subscales of the SIDE could be computed due to too many missing items per subscales or low internal constancy of the subscales. After CIM, data of subscales in the domain of sibling interaction were available for 43–44 siblings pairs, of parental treatment for 41 sibling pairs, of peer characteristics for 38–43 sibling pairs, and of specific events for 35 sibling pairs. Differences in mean values were less than 2% for all imputed variables, and change in internal consistency (Cronbach's alpha) after imputation was only marginal for all subscales except for subscale specific events for which the reliability improved from 0.52 to 0.68. Nevertheless, the reliability of most SIDE subscales before CIM was sufficient and largely comparable with those after CIM.

The amount of missing data on the SIDE was not related to sex, pro-rated IQ, ADHD status, ADHD symptoms, Oppositional behavior, Social and peer problems or Anxious and emotional problems. However, it was negatively associated to children's age (r's between −0.24 and −0.26), indicating that older children had less missing data, which might suggest that the SIDE was more difficult for younger children. All analyses were repeated without implementing data. Though sample sizes were smaller, the results of the repeated measures MANOVA and hierarchical regression analyses were largely comparable. This indicates that the likelihood of a bias due to implementing data is small (Katz 1999). In addition, for reasons of clarity we have specified the sample size for each of the analyses.

Results

Demographic and clinical data

Demographic and clinical data on the ADHD-symptoms and comorbid problem behaviors are presented in Table 1. ADHD probands and unaffected siblings did not differ significantly in age. The difference in age between siblings in the same family (proband minus unaffected) was between −4 and 5 years (mean = −0.51, SD = 2.55). ADHD probands were mainly boys, whereas the unaffected siblings were of an approximately equal numbers of boys and girls. Eighteen sibling pairs (40%) were of opposite sex, 22 pairs were male-male pairs (48.9%) and 5 pairs consisted of two females (11.1%). ADHD probands and unaffected siblings did not differ in pro-rated IQ scores, the difference in pro-rated IQ (proband minus unaffected) was between −26 and 23 (mean = −4.22, SD = 13.32).

Table 1. *Demographic and clinical data*

Dependent measure	ADHD proband			Unaffected sibling			
	M	SD	*N*	M	SD	*N*	*p*
Age, years	13.6	1.53	45	14.1	2.01	45	ns
% Male	84.4		45	53.3		45	< 0.01
Pro-rated IQ scores	98.67	10.64	37	102.89	8.60	37	ns
ADHD symptoms	0.89	0.57	45	−0.89	0.28	45	< 0.001
Oppositional behavior	0.66	0.89	44	−0.66	0.36	44	< 0.001
Social and peer problems	0.42	1.00	44	−0.42	0.64	44	< 0.001
Anxious and emotional problems	0.20	0.94	44	−0.20	0.84	44	< 0.05

ADHD Attention-deficit/hyperactivity disorder; *ns* not significant; ADHD symptoms, oppositional symptoms, social and peer problems and Anxious and emotional symptoms are standardized z-scores of the averaged scores of the respective scales of Conners and SDQ (see text).

Parents rated the ADHD probands as more inattentive, hyperactive and impulsive, as having more oppositional behavior problems, and having more social and peer problems, and as more anxious and emotional.

Differential experiences within discordant sibling pairs

After the proband's scores were reversed, so that positive correlations indicate agreement, statistically significant, positive correlations (r's between 0.30 and 0.57) were found for all SIDE scales (see Table 2). This reflects low to moderate agreement between the siblings on the amount of differences between them.

As can be seen in Table 2, we found significant main effects, with moderate to large effect sizes, indicating differences between ADHD probands and unaffected siblings for three of the four domains of differential environments: sibling interaction, parental treatment and peer characteristics, but not for events specific to the child. For sibling interaction post-hoc analyses showed that sibling antagonism was higher for the probands than for the unaffected siblings. Thus, ADHD probands were described as more antagonistic. For parental treatment, post-hoc analyses indicated that parental control was higher for the ADHD probands, while no significant differences were found for

Table 2. *Means, standard deviations and t-test of sibling pair differences for the 10 SIDE scales*

SIDE factors	ADHD proband M (SD)	Unaffected sibling M (SD)	Pairs			Sibling agreement
			n	*F*	*p*	*r*
Differential sibling interaction, Wilks $\lambda = 0.66$, $p < 0.05$, $\eta_p^2 = 0.34$						
1. Sibling antagonism	2.93 (0.66)	2.52 (0.43)	35	4.42	< 0.05	0.30*
2. Sibling caretaking	2.89 (0.54)	3.16 (0.47)	35	3.37	ns	0.57**
3. Sibling jealousy	3.10 (0.53)	2.74 (0.54)	35	1.10	ns	0.46**
4. Sibling closeness	3.09 (0.66)	3.00 (0.61)	35	1.57	ns	0.34*
Differential parental treatment, Wilks $\lambda = 0.61$ $p < 0.001$, $\eta_p^2 = 0.39$						
5. Parental affection	3.02 (0.23)	2.93 (0.19)	34	2.06	ns	0.50**
6. Parental control	3.22 (0.42)	2.83 (0.41)	34	10.15	< 0.01	0.43**
Differential peer characteristics, Wilks $\lambda = 0.52$, $p < 0.001$, $\eta_p^2 = 0.48$						
7. Peer college orientation	2.81 (0.52)	3.40 (0.50)	30	3.93	ns	0.47*
8. Peer delinquency	3.19 (0.51)	2.77 (0.55)	30	17.80	< 0.001	0.31
9. Peer popularity	3.12 (0.63)	3.19 (0.65)	30	0.17	ns	0.39*
Events specific to the individual, Wilks $\lambda = 0.99$, $p =$ ns, $\eta_p^2 = 0.01$						
10. Specific events	3.07 (0.56)	2.95 (0.36)	28	0.85	ns	0.53**

* $p < 0.05$, ** $p < 0.01$, *ns* not significant.

parental affection. Furthermore, ADHD probands described their peer group as more delinquent.

Differential experiences and differences in parent-rated ADHD symptoms and comorbid behavior

Hierarchical regression analyses indicated that differential sibling interactions were related to differences in both ADHD symptoms and comorbid problem behavior between siblings in the same family after adjustment for differences in age, sex and pro-rated IQ between the siblings. We will discuss the results for each of the four behavioral domains separately.

First, differences in *ADHD symptoms* between the siblings were related to differences in the domain of peer characteristics ($R^2_{\text{change}} = 0.11$, $F_{\text{change}}(1, 27) = 5.09$, $p < 0.05$), as reported by the ADHD proband. More specifically, differences in college orientation (B = 0.52, SE

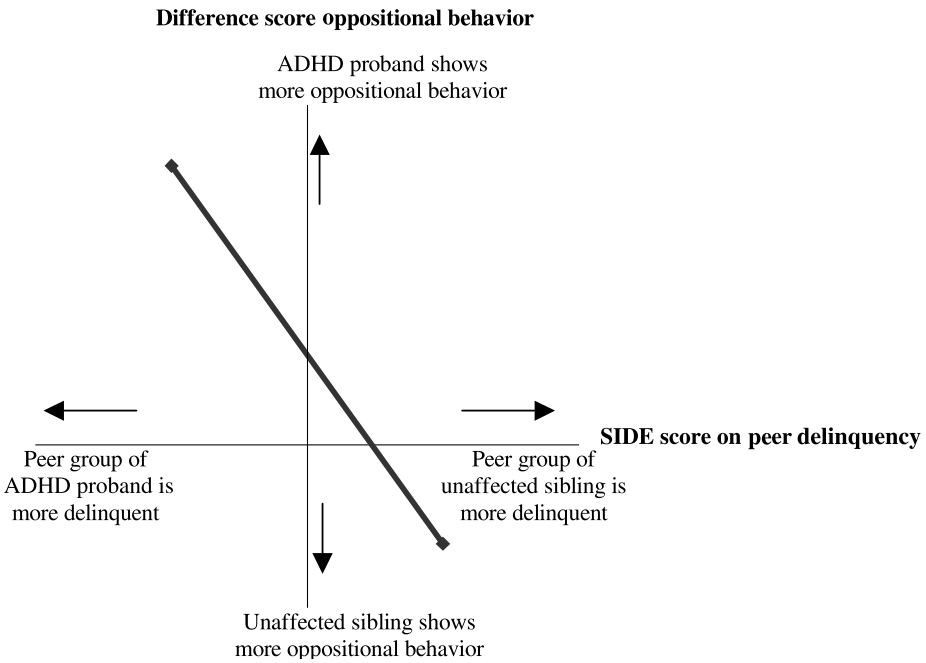

Fig. 2. Main effect of differential Peer delinquency, as reported by the unaffected sibling, as predictor for differences in oppositional behavior, contrasting each ADHD proband with is unaffected sibling. Note: The x-axis represents the SIDE score on the scale Peer delinquency, the y-axis represents the difference in oppositional behavior between the discordant siblings. This figure depicts that the ADHD proband who had a more delinquent peer group in comparison to the unaffected sibling showed more oppositional behavior than his/her unaffected sibling

B = 0.23, $p < 0.05$), as reported by the ADHD proband, were related to differences in ADHD symptoms, indicating that ADHD probands showed more ADHD symptoms than their unaffected siblings, while reporting that the peer group of the ADHD probands were more college orientated.

Second, differences in *oppositional behavior* between siblings were related to differences in the domains sibling interaction ($R^2_{\text{change}} = 0.22, F_{\text{change}}(1.31) = 9.30, p < 0.01$) and peer characteristics ($R^2_{\text{change}} = 0.19, F_{\text{change}}(1.27) = 6.69, p < 0.05$), as reported by the unaffected sibling. When examining the contributions of specific subscales, differences in sibling antagonism (B = −1.13, SE B = 0.37, $p < 0.01$,) and in peer delinquency (B = −0.98, SE B = 0.38, $p < 0.05$) were significant components of the final models, indicating that ADHD probands showed more oppositional behavior than their unaffected siblings, while reporting that the ADHD probands were more antagonistic and the peer group of the ADHD proband were more delinquent (see Fig. 2).

Third, differences in *social and peer problems* were related to differences in parental treatment ($R^2_{\text{change}} = 0.13$, $F_{\text{change}}(1.29) = 5.02, p < 0.05$) and differences in peer characteristics ($R^2_{\text{change}} = 0.12, F_{\text{change}}(1.27) = 4.61$, $p < 0.05$), both with moderate effect sizes. Differences in parental affection (B = −1.99, SE B = 0.89, $p < 0.05$), as reported by the ADHD proband, and differences in peer popularity (B = 0.82, SE B = 0.38, $p < 0.05$), as reported by the unaffected sibling, were significant components of the final models (see Fig. 3). This indicates that ADHD probands showed more social and peer problems than their

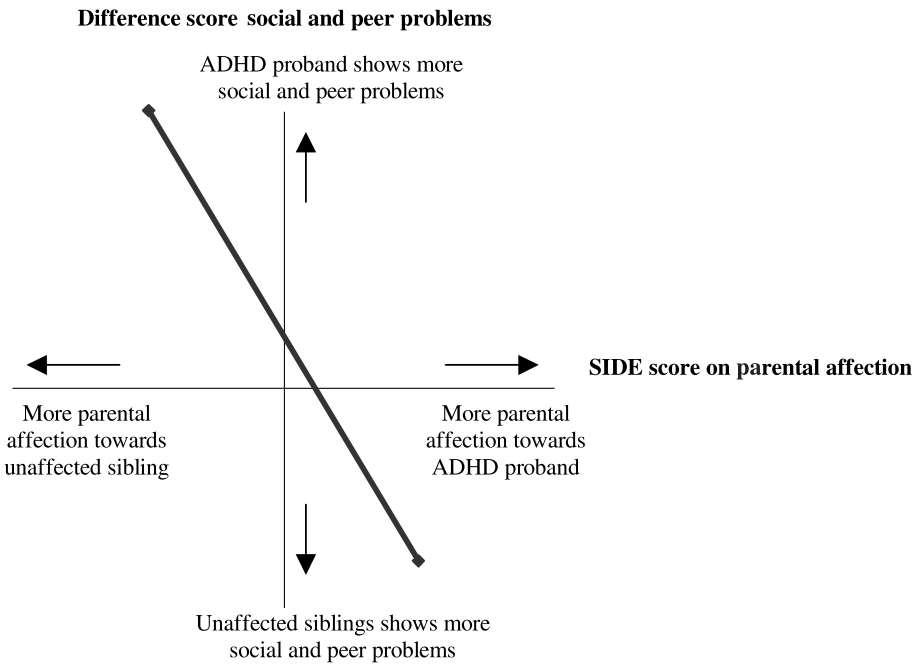

Fig. 3. Main effect of differential Parental affection, as reported by the ADHD proband, as predictor for differences in social and peer problems, contrasting each ADHD proband with is unaffected sibling. Note: The x-axis represents the SIDE score on the scale Parental affection, the y-axis represents the difference in social and peer problems between the discordant siblings. This figure depicts that the ADHD proband who received less affection in comparison to the unaffected sibling showed more social and peer problems than his/her unaffected sibling

Table 3. *Overview of the results of the hierarchical regression analyses*

Side factors	ADHD symptoms		Oppositional behavior		Social and peer problems		Anxious and emotional problems	
Side-informant	ADHD proband	Unaffected sibling	ADHD proband	Unaffected sibling	ADHD proband	Unaffected sibling	ADHD proband	Unaffected sibling
Differential sibling interaction	–	–	–	0.30	–	–	–	–
Differential parental treatment	–	–	–	–	0.17	–	0.23	–
Differential peer characteristics	0.19	–	–	0.25	–	0.17	–	–
Events specific to the individual	–	–	–	–	–	–	–	–

SIDE Sibling inventory of differential experience; *ADHD* attention-deficit/hyperactivity disorder, numbers in the table indicate the effect size for hierarchical multiple regression (f^2), minus-signs indicate that the SIDE domain did not explain additional variance over and above the effects of the differences in age, sex and pro-rated IQ between the discordant sibling pairs.

unaffected siblings, while the ADHD probands received less parental affection and had less popular peer groups than the unaffected siblings.

Fourth, differences in *anxious and emotional problems* were related to differences in parental treatment as reported by the ADHD proband ($R^2_{\text{change}} = 0.15, F_{\text{change}}(1.29) = 6.78$, $p < 0.05$). ADHD probands showed more anxious and emotional problems than their unaffected siblings while reporting that their unaffected siblings received more parental affection (B = −2.27, SE B = 0.87, $p < 0.05$). See Table 3 for a summary of these findings.

Discussion

The present study was designed to test the hypotheses that sibling pairs in the same family, discordant for ADHD, experience differences in the domains of sibling interaction, parental treatment, peer relations and events specific to the individual, and that those within-family experienced differences are related to differences in the siblings' ADHD symptoms and comorbid disorders.

Differences in sibling interaction, parental treatment and peer characteristics

With regard to the *first hypothesis*, and in line with earlier research, ADHD probands and their unaffected siblings reported significant differences in sibling interaction, parental treatment and peer characteristics (e.g., Daniels et al. 1985; Dunn and Plomin 1990; Murphy et al. 2000; Caspi et al. 2004; Liang and Eley 2005). In contrast with a longitudinal study (Liang and Eley 2005), which assessed the association between negative life events and depressive symptoms and included 328 monozygotic (MZ) twin pairs aged 12–19 years, the siblings in our study reported no differences in having experienced specific events. Our findings are in line with a study that investigated the SIDE with siblings discordant for Anorexia Nervosa (Murphy et al. 2000), where only two of eleven items were significantly different between the siblings, which is not more than one would expect by chance.

Differences in sibling interaction, parental treatment and peer characteristics and outcomes

With regard to the *second hypothesis*, specific domains of nonshared environmental differences were significantly related to differences in specific behavior outcomes. This finding is comparable with both epidemiological twin (Wichers et al. 2001; Asbury et al. 2003; Caspi et al. 2004; Liang and Eley 2005; Burt et al. 2006) and (clinical) sibling studies (Daniels et al. 1985; Murphy et al. 2000). We will focus on three main findings.

First, after adjustment for differences in age, sex and pro-rated IQ between the siblings, differences in peer characteristics were related to the severity of ADHD symptoms. One study (Asbury et al. 2003), including 2353 four-year-old MZ twin pairs, found that within-pair differences in parenting were significantly correlated with differences in hyperactivity, conduct problems, prosocial behavior and anxiety. The associations were substantially greater for the extreme 10% of the parenting-discordant and behavior-discordant distributions, as well as in higher risk environments. Discrepancies between this study and the current one may be explained by differences in age of the subjects, sample size, sample composition, or analytic method. In addition, associations in the twin study may have been inflated since parents both reported their own parenting and their children's behavior. Furthermore, differential parenting may have a stronger effect in childhood than in adolescence.

Second, sibling interaction, parental treatment and peer characteristics, but not life-events, were related to differences in oppositional behavior, social and peer problems, and anxious and emotional problems. Below, findings within each domain will be discussed more thoroughly. Earlier studies reported that sibling interaction (i.e. more jealousy and antagonism and less closeness and caretaking) was associated with more shy and internalizing behavior and more emotional anger and externalizing behavior (Daniels et al. 1985; Daniels 1986; Wichers et al. 2001). We only found a association between sibling interaction and externalizing, i.e., oppositional behavior.

Many studies suggest that especially negative parenting behavior directed specifically at each child in the family is associated with prosocial behavior (Asbury et al. 2003), depressive and internalizing symptoms (Reiss et al. 1995; Pike and Plomin 1996; Wichers et al. 2001; Asbury et al. 2003; Liang and Eley 2005) and externalizing symptoms (Reiss et al. 1995; Wichers et al. 2001; Asbury et al. 2003; Caspi et al. 2004; Burt et al. 2006). In addition, differential parenting and differences in behavior correlate stronger when the behavior between the sibling is more discordant and in and higher risk environments (Asbury et al. 2003). Our findings were partly in line with these studies: we did find a relationship between differential parental treatment and social and peer problems as well as anxious and emotional problems, but not with oppositional behavior. In addition, we found stronger associations for positive parenting, i.e., affection.

Differences in peer characteristics were generally related to both more ADHD symptoms and more comorbid problem behaviors of the ADHD probands. Earlier studies, which concur with our study, reported peer characteristics to be related to depression and internalizing symptoms (Wichers et al. 2001) and externalizing behavior such as conduct problems (Carbonneau et al. 2002). In accordance with relatively recent reviews (Harris 1998; Turkheimer and Waldron 2000), children's and adolescents' development is not so much determined by parental influences as by the peer group; peers are the primary context for the development.

We did not find an association between specific events and the different dimensions of problem behavior. Earlier studies did find such an association: the more events impacted more specifically on the proband, relative to the other sibling, the more internalizing, depressive and externalizing psychopathology he or she was likely to display (Wichers et al. 2001; Liang and Eley 2005). This inconsistency may be explained by differences in measurements (Liang and Eley 2005) and informants (Wichers et al. 2001).

Third, consistent with other studies (Daniels and Plomin 1985; Pike et al. 2000), low to moderate agreement between siblings on differential experiences was found as evidenced by bivariate correlations between the ratings on the SIDE, and no similar pattern for probands and unaffected siblings was found in the multivariate analyses. A possible explanation for this finding is that ADHD children may have a bias in their social self-perceptions. Evidence suggests that, when ADHD boys' perceptions is compared directly to those of their parents, ADHD boys' reports are positively enhanced relative to those of control parent-child dyads (Gerdes et al. 2003). This finding is in accordance with the cumulating evidence for positive illusory self-perceptions in children with ADHD (Hoza et al. 2004). The mechanisms underlying the positive illusions in children with ADHD might be related to a self-protective function in coping with deficits in the social domains (Hoza et al. 2002).

Limitations

Our data are cross-sectional, and do not permit separating cause and effect in the relationship between differential sibling experiences and sibling behavior. It is likely that children and parents affect each other in a bidirectional way: children shape their environments ((re)active gene-environment correlations). Further, different environments of siblings may be confounded with genetic differences between the siblings (Caspi et al. 2004), which can lead them to experience their environment differently (Carbonneau et al. 2002). Therefore, this study should be complemented by a longitudinal design that controls for genetic influences.

As Rowe (1994) noted, caution is warranted in drawing conclusions about strength of specific nonshared influences. Developmental processes are much more complex than the mathematical models used to test hypotheses. For instance, effects of developmental processes are usually not restricted to the variables tested. Furthermore, the family system is not isolated from the rest of the world. Parental nonshared influence might be shared influence relative to the general population.

Another limitation is the possible inflation of type I and II errors. First, the number of type I errors may be inflated due to multiple testing. However, we found 6 of the 32 regression analyses to be significant (with f^2 between 0.17 and 30) which is significantly more than what would be expected by chance ($n = 32$, test proportion 0.05, $p <$ 0.01 by binomial test). Second, type II error may be inflated by the small sample size. Nevertheless, post-hoc power analyses showed us that this sample gave us a statistical power of 62–89% to detect medium to large effect sizes.

Due to the selection of the sample, the range of comorbid symptoms was larger than the range of ADHD symptoms. This increased the likelihood for finding more effects for comorbid behavior than for ADHD symptoms. Last, in this study all subjects were Caucasian European and the probands were mainly males with combined type of ADHD. It is unclear how generalizable the results are to other populations.

Clinical implications

Nonshared environmental influences like differential sibling interactions, differential parental treatment and differences in peer characteristics seem particularly salient in explaining severity and comorbidity in children with ADHD. The clinical application of these findings would suggest that prevention or treatment of children with ADHD should place considerable focus on these nonshared environmental influences. These results add to the notion that it is important not only to assess the affected child but the entire family (Pike and Plomin 1996). Especially the information of an unaffected sibling with regard to nonshared environmental influences within that family might provide useful additional information for attunement of intervention of prevention strategies.

Appendix

Internal consistency and sample items for each of the SIDE subscales

Sibling interaction

Sibling Antagonism (9 items, $\alpha = 0.72$) – ''In general, who has been more likely to show feelings of anger toward the other?''
Sibling Caretaking (6 items, $\alpha = 0.44$) – ''In general, who has been more likely to take responsibility for the other?''
Sibling Jealousy (6 items, $\alpha = 0.63$) – ''In general, who has been more likely to get jealous of the other?''
Sibling Closeness (3 items, $\alpha = 0.57$) – ''In general, who has shown more affection toward the other?''

Parental treatment

Parental affection (10 items, $\alpha = 0.57$) – ''mother/father has shown interest in the things we have done''.
Parental control 10 items, $\alpha = 0.83$) – ''mother/father has disciplined us''.

Peer group characteristics

Peer college-orientation (12 items, $\alpha = 0.86$) – ''ambitious'', ''achieving in school''.
Peer delinquency (7 items, $\alpha = 0.72$) – ''rebellious'', ''bad group''.
Peer popularity (6 items, $\alpha = 0.70$) – ''outgoing'', ''achieving status in social situation''.

Events specific to the child (13 items, $\alpha = 0.68$)
''Who has been more influenced by an extraordinaire event?''
''Who has been more influenced by teachers in school?''

Note: Items 48 and 66 of the original SIDE were deleted to improve reliability of the scale ''Peer delinquency'' and the domain/scale ''Events specific to the child'', respectively.

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